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Respiratory Symptoms and Peak Flow Associated with Indoor and Outdoor Air Pollutants in the Southwest

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A symptom-stratified, geographic cluster sample of 117 middle class households was studied. Symptom daily diaries and peak flows were obtained for 211 subjects over a two-year period. Indoor sampling in a sample of houses was performed for O_3, TSP, RSP, CO, temperature (*T*), and relative humidity (*RH*). Questionnaires determined type of stove and number of smokers in all households. Ambient pollutants (O_3, TSP, CO, NO_2), were monitored in or near the clusters, as were *T* and *RH*. Smoking in the household was significantly correlated with TSP and RSP. Indoor CO was significantly correlated with gas stove usage. Normal young adults under age 25 had daily peak flows (PEF) associated with outdoor O_3 after adjusting for other factors). Asthmatics' PEF was associated with smoking, gas stove use and outdoor NO_2, and with outdoor O_3 and temperature, after controlling for other factors. Indoor and outdoor factors affected asthmatic symptoms, after controlling for age, sex, smoking and other ambient environmental variables.

Recent reviews[1–3] have highlighted the growing importance of indoor pollutants as risks for respiratory illness. One major area of concern and controversy is involuntary cigarette smoke, especially its effects on children.[4,5] Another area of concern has been the effects of emissions from gas stoves in the home.[6–8] In large part, the drive for energy efficiency, to "tighten" the home environmentally, leads to much less infiltration and exfiltration, allowing pollutant concentrations to increase.[1] In addition, many new housing construction activities, such as the use of particle board and plywood, have increased levels of other gases, such as formaldehyde, such that they have the potential to produce effects on health.[9]

The accurate determination of the health effects associated with ambient pollutants, considered in part by the National Ambient Air Quality Standards, is another important consideration. As people spend most of their time indoors (75–90%), they are exposed more to indoor and less to outdoor pollutants.[1] This may indicate different exposure-response relations. Further, the nonuniformity of indoor/outdoor exposures implies different sets of indoor-outdoor exposure mixes. Thus, the relative contributions to effects on health of indoor and outdoor pollutants, and their interactions, need to be determined.

Nitrogen oxides (NO_x) and carbon monoxide (CO) are often found indoors. Studies of gas stove usage have demonstrated that levels of NO_x are dependent on combustion efficiency and ventilation, and indoor concentrations are often higher than those outdoors.[1,10] The impact of environmental tobacco smoke on indoor concentrations of NO_2 is usually small compared to the impact of gas stoves.[10] In addition, residential wood and coal stoves are potential sources of CO and NO_x as well as respirable particles.[1]

On the other hand, ozone occurs predominantly outdoors. Nevertheless, studies on acute effects on pulmonary function in children and adolescents[11–16] indicate a consistent finding that as outdoor ozone increases, peak flow and spirometric flow volume measures decrease in a fairly linear fashion over the range of values studied (0.01–0.15 ppm), even after controlling for temperature and other pollutants. These results are consistent with controlled human exposure studies.[17] Exercise and the contribution of other pollutants and temperature appear important for the effects seen.

This paper examines daily respiratory symptoms and peak expiratory flows (PEF) in children, young adults and adults; many of the adults were chosen because they have prior respiratory conditions. It examines the influence of indoor gas use and particulate pollutants, of outdoor pollutants, and meteorological phenomena on respiratory status. This study was conducted in a representative community population sample in Tucson, Arizona.[18] Previous studies in this population sample indicated the presence of relationships between weekly symptoms and both air pollutants and allergens, controlling for weather.[19] Prior analyses of the present data base evaluated some effects of biological agents (algae, fungi, pollen), suspended particulate matter,[20] and environmental tobacco smoke.[21,22] This paper reports on more recent findings from this study.

Methods

A total of 117 households (229 subjects) in Tucson were derived from a stratified sample of households in four geographic clusters from a representative community population under study.[18] The families studied were middle class; they represented families with and without certain reported symptoms and diagnoses (asthma, allergies, airway obstruction). They were monitored over a two-year period. Daily diaries provided symptom information. Peak flows (per-

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formed between 1500 and 1900 hours each daily) were obtained using a mini-Wright peak flow meter,[23-26] which provides results in arbitrary, relative numbers (rather than in clinical, absolute terms). The mini-Wright peak flow meters were calibrated monthly. The subjects were trained in their use by trained nurses, and monthly phone checks and seasonal visits were made to ensure proper usage. Participation of families from week to week was consistent, except for random one to two week intervals of absence (related to vacations, holidays, family crises). Individuals were absent on occasion due to incapacitating illness as well. Because family members fell into different subgroups, their absence affected the subgroup denominators differently, though randomly. There were sufficient diary responses per day (person-days, the denominator each day >5) for a majority of days in each of the seasons; the average participation per day in each symptom subgroup was 10.2–14.6 (Table I). Symptom rates per 100 person-days were calculated for each day from daily diary data within symptom groups and within exposure groups, for comparative purposes.

There were 24 in a young cohort (5–25 years) who were studied over an 11-month period in 1979–1980. After age and height correction, there was no difference between individuals in baseline peak flows (by ANOVA); all were within the normal range (within one standard deviation of 100% predicted). To remove effects of age and sex, and to avoid use of arbitrary absolute numbers, each daily PEF was converted into a standard deviation score (z-score), utilizing means and standard deviations [$z = (x_i - \bar{x})/s$] of each individual's values. Thus, values each day were deviations from individual means and all values were in the same units (with mean 0 and standard deviation 1). Average daily variations observed were compared to day to day variations in PEF in normal individuals.[23]

There were 23 adult asthmatics in one of the geographic clusters, and a total of 22 other adult asthmatics, with daily peak flows. There were 321 and 354 days of observation, respectively, with sufficient information (>5 individuals/day) on these asthmatic groups. Person-days of observation for asthmatics and for other syndrome groups are shown in Table I. For adults, standard deviation scores (z-scores) of peak flows used sex-age-height specific values. Thus, all PEF were adjusted for anthropomorphic covariables, and syndrome groups retained their relative status in regard to pulmonary function (i.e., those with a syndrome like asthma, AOD have lower pulmonary function). Symptom rates were seasonal,[20] so season was adjusted as well, using z-scores.

Information about the characteristics of the house, yard and streets, heating, cooling, type of stove, other appliances, and smoking in the household, were obtained from all households. As reported elsewhere[20,27,28] micro-indoor and outdoor monitoring (i.e., in and around the residence) have been conducted in a random cluster sample representative of all study households for temperature, humidity, air pollutants and aeroallergens (pollen, bacilli, fungi, and algae). Indoor total and respirable (>2.5 μm) suspended particle (TSP and RSP) levels were related to environmental tobacco smoke but not gas stove usage, while CO levels were the reverse.[21] Macro-monitoring for air pollutants and pollen were conducted simultaneously in the center of each of the geographic clusters, and regional air pollutant and meteorological data were provided.

Statistical analysis of daily relationships between environmental variables and respiratory responses used SPSS programs on a DEC 10-Cyber 175 computer system. Multifactorial analyses of variance (ANOVA) and regression methods were utilized to examine interactions and control for colinear variables. In the ANOVAs, regression options produced multiple analysis of covariance solutions also. Metric and nonmetric covariates were used to remove variation related to them before analyzing the independent variables. All results are presented after controlling for other variables. Reported analyses usually have days as the unit of observation; some use person-days as the unit of observation. Person-days represent the denominator for each day and, thus, the sample size (Table I). When person-days were the unit of study, a person-days dummy variable was used in analysis to eliminate effects of the different number of individual person-days of observation. Unless otherwise noted, statistical significance refers to a $p < 0.05$.

Table I. Sample size in the adult syndrome groups.

Symptom groups	No. of persons[a]	No. of person days	Mean daily denominator[d]
Asthmatics	45[b]	6182	10.2
AOD S2 groups[c]	68	8545	14.0
Allergic subjects	62	8440	14.6
Asymptomatic subjects	29	3663	7.4

[a] Only those with adequate data.
[b] 23 in the asthma cluster and 22 in other clusters.
[c] Airway obstructive disease symptoms.
[d] More than 6 persons/day.

Results

Indoor pollutant levels found in this study have been described previously.[20,21,27] Briefly, indoor TSP had a maximum of 68.5 μg/m³, while simultaneous micro-outdoor TSP reached 169.6 μg/m³. RSP indoors reached 49.7 and outdoors 124.5 μg/m³. The type of dust found inside, on the whole, could not be identified (using electron microscopy). Indoor CO was quite low (below 2.4 ppm) and micro-outdoor CO was below 3.8 ppm; CO correlated significantly with the presence of gas stoves. The presence of gas stoves was used as the variable qualitatively replacing NO_2 (since NO_2 measurements were not available). Neither gas stove usage nor household smoking was associated with social status (measured by education, income, occupation, crowding) in this sample. Indoor ozone (O_3) was very low indoors (measured as between 0 and 0.035 ppm). Pollen algae and fungi sampling results have been described previously.[20] There was no relationship between work exposures and indoor TSP or RSP; thus, no noticeable paraoccupational exposure was present.

Peak Flows in the Young

In the younger age group, more time was spent outdoors. Average daily PEF was shown to be significantly related to environmental factors (by multifactorial ANOVA, $F = 2.97$, $df = 29$ and 644, $p < 0.001$).

Further analyses were employed to examine the direct relation of O_3 to PEF. PEF was adjusted by a multiple regression for person-days, TSP, temperature and stove use ($R = 0.23$, $p < 0.001$); the other environmental factors had significant interactions with PEF. The regression of adjusted PEF on O_3 was linear with adjusted PEF = 0.287 − 6.025 O_3 ($n = 674$ person days, $p = 0.013$); the intercept and slope were both statistically significant ($p < 0.001$). Residual analysis of PEF with O_3 was significant as well (residual PEF = 0.17 − 3.20 O_3; intercept and slope statistically significant). After removing TSP (because of smaller n) and adjusting PEF for relative humidity as well, O_3 and adjusted PEF had an $R = -0.56$ ($p < 0.0001$, $n = 1513$ person days, adjusted PEF = 0.17 − 3.87 O_3); residual PEF was significantly related to O_3 as well ($p < 0.002$). Gas stove use was of borderline significance in the first regression ($p = 0.066$), but not significant ($p < 0.25$) in the second analysis.

Adults' Peak Flows

In adults, smoking had the biggest effect on PEF in every syndrome group. In normals and all allergic subjects, there were no consistent trends.

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Table II. Peak flows standard deviation scores in asthmatics (asthma cluster) by outdoor ozone and temperature interaction.[a]

Average maximum hourly	Maximum temperature (°F)		
O_3 (ppm)	<61	61–80	80–96
<0.038	−1.94	−1.85	−2.17
	(217)[b]	(219)[b]	(174)[b]
0.038–0.051	−1.99	−1.78	−1.75
	(327)[b]	(490)[b]	(388)[b]
0.052+	−2.11	−1.71	−1.57
	(30)[b]	(549)[b]	(794)[b]

[a] ANOVA $F(8,312) = 9.25$, $p < 0.0001$ ($R^2 = 19.2\%$).
[b] Person-days of observation.

In 23 adult asthmatics in one geographic cluster, where indoor monitoring was most complete, PEF was significantly related to humidity indoor TSP, and active smoking. After adjusting for these other variables, PEF was significantly related to the interaction of O_3 and temperature; high temperature had an effect when O_3 was low and there was a nonsignificant trend of O_3 with PEF only in low temperature (Table II). Also, those in houses with low to moderate micropollen had a higher average PEF (z-score mean of −1.18) compared to those with high levels ($\bar{z} = -2.51$, ANOVA, $F = 197.5$, $p < 0.0001$).

Asthmatics who spent 4–8 hours outdoors per day had significantly lower PEF than those with less than four hours outdoors per day (t_3, $p < 0.05$). In all asthmatics, gas stoves were associated with a significantly less average PEF (average z-score = −2.16) compared to average PEF in those with electric stoves (average z-score = −0.76, $p < 0.001$), using all person-days of observation. This held up within smoking groups as well, though the difference was greater in current smokers. (This relationship with NO_2 and gas stoves was found in allergic and normal smokers as well.) There was a significant interaction of type of stove and outdoor NO_2 with PEF. On the days when there was measurable high outdoor NO_2, it was significantly related to decreased PEF only in those with electric stoves (Table III). Those with gas stoves had significantly lower PEF than those with electric stoves when outdoor NO_2 was low.

In adults with the AOD syndrome, the overall relation of environmental variables with PEF was highly significant by multifactorial ANOVA, $F = 3.84$, $df = 15$ and 243, $p < 0.001$). PEF was adjusted for the significant dependent effects of smoking, relative humidity, indoor TSP, and gas stove usage; the significant effect of O_3 was seen when TSP was ≤ 76 $\mu g/m^3$. As there is a negative O_3-TSP correlation in this subset of days, related to their peak occurence in different times of the year, the TSP-PEF relation was paradoxical. When these other variables were controlled in multiple regression analysis, the regression coefficient for O_3 was −5.946 ($p < 0.005$ by t test); the regression coefficient for TSP was +0.004. Since the number of days with TSP was less than half of the days with O_3 (260 vs. 586), PEV adjusted for the other variables, ignoring TSP, was related to O_3 (Table IV). The decrease in PEF on days with $O_3 > 0.051$ ppm is significantly different than normal day to day variation.[23]

Table III. Interactive effect of gas stove usage and outdoor NO_2 on adult asthmatic average $\dot{V}max$ adjusted for other effects.[a]

NO_2 (ppm)[b]	Stove	
	Gas	Electric
≤0.264	−1.54[c]	−1.24[c]
0.027–0.0385	−1.53	−1.51
0.039+	−1.53	−1.65

[a] ANOVA F 15,349 = 8.7, $p < 0.0001$.
[b] Distribution of days divided into equal thirds.
[c] t-test significant, $p < 0.01$.

Symptoms

The average daily prevalence rate for the presence of any symptom was 42.8%. Season specific prevalence rates ranged from 1.0 to 19.1% for eye irritation and 4.14–64.7% for rhinitis. The normal young cohort and asymptomatic adults ("normals") had no significant relations between symptoms and environmental factors.

"Asthma" symptoms ("attacks," wheeze, dyspnea) had significantly higher correlations with increased use of medication and increased physician visits, but their attacks were not day-of-week dependent in this study.

Temperature and precipitation had a significant interaction which was related to reported asthma symptoms in these asthmatics, as shown in Table V. (These individuals, affected also by outdoor O_3 in high temperatures, spent more time outdoors than the average.)

Table IV. Relation of adjusted daily averge peak flows (standard deviation scores) with outdoor O_3 in adults with the AOD syndrome.[a]

	Average maximum hourly O_3 (ppm)[c]		
Adjusted daily[b]	<0.038	0.0038–0.051	0.052–0.12
Average PEF	−0.581	−0.582	−0.801

[a] ANOVA, $p < 0.0001$ ($R^2 = 4.2\%$), n(days) = 586.
[b] Adjusted for other significant environmental covariables.
[c] Distribution divided into equal thirds.

Daily prevalence rates of productive cough in asthmatics (average of 33.2%), were corrected for age, sex, smoking, temperature, and relative humidity (all shown previously to be independently significant). The corrected rates were associated with high outdoor CO and NO_2 (occurring in winter) and gas stove usage [ANOVA, $F(15,305) = 8.96$, $p < 0.001$].

In allergic subjects, outdoor NO_2 was associated with increased age-sex-smoking adjusted rates of eye irritation in moderate to high temperatures [ANOVA, $F(5,493) = 24.92$, $p < 0.001$]. The prevalance rates progressed from 14.6% to 20% to 28.8% with increasing NO_2 (Table III).

Discussion

Subgroup division was necessary because subgroups responded differently to environmental stimuli (as shown herein). This created smaller daily denominators. Participation rates were equally distributed within and between subgroups. Further, individuals with greater daily symptomatology were not significantly over-represented in the denominators. Thus, differential participation was not a major source of bias in these analyses.

The statistical independence of daily values has been a major concern in statistical analyses of these types of data. Generally, the problem relates to the day-to-day autocorrelation in pulmonary function measures and prevalence rates. Pulmonary function is relatively stable from day to day,[23] a known biological necessity of life. Thus, one can only analyze deviations from an individual's or a group's average daily value or regression line (e.g., residuals) to reflect changes. Using multiple linear or logistic regressions does not change this phenomenon. Further, deviation values are likely to have less auto regression (unpublished data) and be distributed more closely to a Gaussian distribution.[15]

Incidence rates of symptoms would be independent, but the critical ones, such as asthma attacks/symptoms, occur infrequently; there were only 75 incidence days in 3820 person-days of observation. The prevalence of asthma attacks-

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Table V. Prevalence rates (%) of daily "asthma attack" symptoms in asthmatics (asthma cluster).[a]

Precipitation	Temperature[b] Low	Mod	High	Total	(No. of days)
None or low	2.45	0.41		0.65	(276)
High	5.91	0.62		0.38	(45)
Total	3.56	0.84	0.10	0.89	(321)

[a] ANOVA $F = 21.2$, $p < 0.0001$, $R^2 = 26\%$.
[b] Divided into equal thirds.

/symptoms are not independent in an individual from day to day, so deviations of daily rates in a group cannot be said to be strictly independent. However, our data show that asthmatics differ greatly in their components of variance and in their contributions to variance measures over time. Thus, combining coefficients from individual multiple logistics is medically meaningless and may be incorrect statistically. The use of ANOVA for repeated measures is a promising solution to the problem; it allows one to evaluate interactions.[29] Using deviations from solutions of models fitted to the data decrease autocorrelations significantly and produce variates that are more normally distributed.[15,19] The use of the two approaches together is the approach used in the analyses presented in this paper.

Those under age 25 in this study were found to spend more time outdoors and have more exercise than adults. Thus, the previous[21,22] and present findings of an outdoor O_3 association with PEF after controlling for other variables is not surprising; it clarifies and confirms our previous studies[14] and those of Lippmann *et al.*[16] The interactive effects of O_3 with outdoor TSP and with temperature[21] confirmed prior suspicions as well. However, the O_3 level at which effects were seen, though consistent, is found in a range of O_3 lower than expected (0.052–0.08 ppm) as well as in the higher range (0.08–0.12 ppm). It is conceivable that O_3 is acting as a surrogate variable. However, in the adult asthmatics, this type of analysis effectively eliminated the association of PEF and "attacks" with O_3. The incidence rates of asthma attacks in asthmatics, based on 75 in the numerators, ranged from 2.5 to 3.8% with increasing O_3 levels, but this was not statistically significant. Surprisingly, there were no effects on symptoms noted, indicating a greater sensitivity to change of the PEF measures.[16,23–26] The same statement can be made for normal adults in this study, whose PEF was affected by outdoor NO_2 and O_3.

Recently, studies of the effects of gas stove usage have been performed by several groups,[6,7] where NO_2 was measured and passive smoking was studied as well. The results have been quite contradictory in these studies and others.[6–8,30,31] The effect of the presence of gas stoves and indoor CO (a surrogate and correlate of gas stoves) were seen in sensitive individuals. Their relations with PEV were consistent[21,22] especially in asthmatics, and not unexpected.[1] Outdoor NO_2 had such effects also. The presence of gas stoves (and/or indoor CO) was clearly related to irritant responses in adults (eye irritation, sore throat, rhinitis, peak flows).[21,22] However, gas stove usage often interacted with outdoor pollutants or temperature in relation to many of the symptoms, and these were sometimes paradoxical.[21,22] Thus, further studies of measured gas stove emissions, in conjunction with measures of other pollutants, are necessary.[41,42] The interaction of active smoking, gas stove usage, and outdoor NO_2 in relation to PEF were more interesting. Unfortunately, one cannot say definitively what indoor pollutants are truly represented in this sample because of limited measurements; minor perturbations of other unmeasured pollutants may be involved also. A major interest is whether the carboxyhemoglobin and methemoglobin (from NO_2 and NO) accumulate from exposure to multiple sources (especially smoking and gas stove emissions) and act in an additive fashion as adverse risks to health, as implied in animals.[32] One could estimate that gaseous pollutants effects would be seen from environmental tobacco smoke (ETS) if its concentration were at least 3–5 times greater, the approximate degree that stove emissions exceed those of sidestream smoke,[1] or if it were nine times greater to see the same effects as active smoking, based on dose estimates of sidestream smoke.[33]

Utilizing indoor measurements showed that indoor levels of total and respirable particles were high in homes with smokers.[27] However, there was no direct effect of ETS on children's symptoms or lung function. Indoor TSP had an effect on daily symptoms (eye irritation, productive cough) in all adults and on asthmatics' symptoms and PEF.[21,22] However, since ETS did not,[21,22] the relationships must be associated with other characteristics of the indoor TSP.

ETS is definitely responsible for annoyance and sensory irritant effects.[1–5,22] However, the specific contributions of sidestream smoke to effects of personal exposures have not been sufficiently documented, and health effects in children and adults, sensitive and not, have to be clarified.[22] Two studies of the effects of passive smoking on asthmatics and controls have demonstrated conflicting findings.[34,35] We had not previously found direct effects of passive smoking in children's or adults' lung function,[21,35] nor did we in this study. Indirectly, the relationship of O_3 with PEF seen mainly in children who were not passive smokers[22] may indicate some blunting effect of passive smoking, similar to the lessened sensitivity of smokers to O_3 found in controlled human exposure studies.[37–40]

There have been very few measurements of the extent or behavior of pollen or other microorganisms indoors. In our study, algae found outside or in evaporative coolers were not found indoors. They did not affect daily symptoms or peak flow.[2] Fungi were either ubiquitous or only found in one location (indoors, outdoors, or in coolers). (Aspergillus found indoors does not appear to produce skin test reactivity.) In preliminary analysis, they appeared to be related to symptomatology,[20] but the present multivariate analyses indicated they had no negative effects when other host and environmental conditions were taken into account. On the other hand, micro-pollen affected peak flow in sensitives, asthma attacks/symptoms and other symptoms in the various adult groups in this study[21,22] and in a previous study.[19] Thus, micro-pollen should be studied further as a significant environmental health phenomenon. Other aeroallergens deserve further study as well, such as cat/dog dander; pets were significantly related to asthma symptoms in our study.

In this study, sensitive groups (children, asthmatics, allergics, AOD) had more reactions to indoor and outdoor pollutants. Asthmatics appear to respond to most of the environmental factors considered. Indoor pollutants affected sensitive adults as did outdoor pollutants, and the effects of the indoor pollutant is related to the amount of time spent indoors. Unfortunately, because of sample size and the number of confounding factors, a precise exposure-effect relationship could not be determined. This would appear to be a major methodological problem for this type of study, as one must account for covariables (age, sex, smoking), symptoms (asthma, allergy, AOD), indoor and outdoor pollutant and pollen differences. Further, it is not inconceivable that O_3 or gas stoves (as examples) are acting as surrogates or indexes of some unknown factors which were not measured. Other indoor exposures were not considered in this study. Speciation of particulates may be critical as well, though not done in this study. We found that many factors related to indoor characteristics affect the strengths of the pollutants: the number of smokers in the home, the type of cooking system, fuel, the nature and volume of road surfaces, and meteorological factors. Personal exposure variables, as they indicate that subjects experience pollution levels very different from those measured at a nearby fixed monitoring station, and even different from those measured indoors,[41,42] should be

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pursued further to obtain total exposure estimates in this type of epidemiological study.

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